A DISSERTATION ON

"OCCURRENCE OF INTRACAVITARY THROMBUS IN THROMBOLYTIC ERA"

M.D DEGREE

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CERTIFICATE

This is to certify that the dissertation titled ***"STUDY OF OCCURRENCE OF INTRACAVITARY THROMBUS IN THROMBOLYTIC ERA"*** *submitted by* ***Dr. R.V. SEBASAN*** *to the Faculty of General Medicine, The Tamilnadu Dr. M.G.R. Medical university, Chennai in partial fulfillment of the requirement for the award of M.D. Degree Branch I (General Medicine) is a bonafide research work carried out by him under our direct supervision and guidance.*

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DECLARATION

I, **Dr. R.V. SEBASAN,** *solemnly declare that the dissertation titled* ***"STUDY OF OCCURRENCE OF INTRACAVITARY THROMBUS IN THROMBOLYTIC ERA"*** *has been prepared by me. I also declare, this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University, board either in India or abroad.*

This is submitted to the ***Tamilnadu Dr. M.G.R. Medical University****, Chennai in partial fulfillment of the rules and regulations for the M.D. Degree Examination in General Medicine to be held in March 2007.*

Place : Madurai

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CONTENTS

GLOSSARY

AWMI	-	Anterior wall myocardial infarction
CAD	-	Coronary artery disease
DM	-	Diabetes Mellitus
ESV	-	End Systolic Volume
EDV	-	End Diastolic Volume
LVEF	-	Left Ventricular Ejection Fraction
LVIDs	-	Left Ventricular Internal diameter in systole
LVIDd	-	Left Ventricular Internal diameter in diastole
LV Thrombus	-	Left Ventricular Thrombus
MR	-	Mitral Regurgitation
FS	-	Fractional Shortening
SHT	-	Systemic Hypertension
WMSI	-	Wall Motion Score Index
RWMA	-	Regional Wall Motion Abnormality
STEMI	-	ST Elevation Myocardial Infarction
ICCU	-	Intensive Coronary Care Unit
Echo B	-	Baseline Echo
Echo 14	-	Echo on Day 14
Echo 28	-	Echo on Day 28

INTRODUCTION

Left ventricular thrombus is common after acute myocardial infarction; in different studies, the incidence of left ventricular thrombi has been reported varying from 28% to 54 %. **(1-9)**With the advent of systemic thrombolysis as standard treatment in acute myocardial infarction, few studies reported the effects of thrombolytic agents on ventricular thrombi, showing that thrombolytic therapy reduces their frequency**. (10-13)**

However the impact of thrombolysis on the incidence of LV thrombi has been incompletely investigated. In this study, a wide patient population with STEMI was evaluated through serial echo to identify whether any significant association does exist between wall motion abnormalities and thrombus formation. The influence of co-morbid factors (DM, AGE, SEX, SMOKING, SYSTEMIC HYPERTENSION, DYSLIPIDEMIA, FAMILY HISTORY OF CAD) were also evaluated as possible predictors of LV clot formation.

REVIEW OF LITERATURE

Left ventricular thrombus commonly follows an acute myocardial infarction, particularly when it involves a large area of myocardium of the apex and anterior wall. The main clinical implication of left ventricular thrombus is related to possible embolic complications, most importantly affecting the cerebral circulation, that deleteriously influence the clinical outcome of approximately 10% of patients surviving the acute phase of an infarction.

The Diagnosis of Thrombus:

The diagnosis of thrombus is based on the *detection of an* ***echo dense mass with defined margins, clearly identifiable throughout the cardiac cycle, adjacent to asynergic Myocardium, visible in at least two different echo Cardiographic views and distinguishable from other Cardiac structures or artifacts*** (Asinger et al 1981a, Stratton et al 1982, Visser et al 1983a, Weinreich et al 1984). Detection of adjacent myocardial asynergy is a necessary precondition when diagnosing left Ventricular thrombus following an acute Myocardial infarction (Asinger et al 1981b). The thrombi are located

most frequently but not exclusively in the left ventricular apex (Asinger et al 1981a, Stratton et al 1982, Visser et al 1983a, 1985). The presence of apical dilatation or dyskinesia and the greater degree of Myocardial necrosis which more frequently characterizes an anterior as opposed to an inferior infarction predisposes to a greater focal stasis of intra-cavitary blood and to the development of thrombus (Yater et al 1951, Jordan et al 1952, Asinger et al 1981b,); Consequently, most thrombi develop in the left ventricular apex of patients with anterior myocardial infarction.

In addition, normal and pathological structures present near the left ventricular apex must be differentiated from thrombus, The recognition of papillary muscles can be aided by identifying them in their usual site and excluding wall motion abnormalities. Muscle trabeculae are often associated with left ventricular hypertrophy and may appear as muscular bridges across the ventricular cavity. The detection of an echo-free space on each side of this structure may exclude this mis-interpretation. Chordal structures (true chordae tendineae or chordal-like structures) can be distinguished from thrombus by the absence of adjacent asynergic myocardium, the

detection of an echo-free area on each side of them and their normal straightening motion during diastole. Finally, when examining the left ventricular cavity, an incorrect tangential visualization of the apex simulating the presence of a thrombus must be avoided.

Echocardiographic evaluation of the cardiac apex should be obtained with the patient in the lateral decubitus position and the transducer placed at the point of maximal apical impulse. Slight changes in transducer position may be necessary for a complete examination of the cardiac apex, whose geometry can be altered by an aneurysmatic dilatation or a dyskinetic motion.

At present, the sensitivity and specificity of echocardiography in the diagnosis of left ventricular thrombus can be considered as being approximately 95 and 88%, respectively (Ezekowitz et al 1982, Stratton et al 1982, Visser et al 1983a, Domenicucci et al 1987.)

Inspite of correct methodology and a thorough echo search for thrombus, false negative diagnosis may occur. These are normally attributable to the small dimension of the intracardiac mass; **a thin,**

layered thrombus with a maximum thickness of less than 0.6cm will not be identified (Mikell et al 1982a)

Other Diagnostic Techniques:

With regard to other techniques proposed for the evaluation of left ventricular thrombus **Indium 111 platelet scintigraphy** should be considered complementary to echo, since it can detect platelet turnover on the thrombus surface and appears suitable to assess the effects of anti-thrombotic drugs(Ezekowitz et al 1982);however, inspite of its high specificity this method is not cost effective and is time consuming, in addition it has a low sensitivity, being unable to show hematologically inactive thrombi. The low feasibility and repeatability and the high cost of **CT and MRI** (notwithstanding their good diagnostic accuracy), make these methods advisable only when initial echo studies are technically inadequate or equivocal.

PATHOGENETIC DETERMINANTS OF THROMBUS FORMATION

From the initial observation that thrombus is always associated with left ventricular wall motion abnormalities (Asinger et al 1981b), further studies (Stratton et al 1982, Visser et al 1983b, Weinreich et al

1984, Domenicucci et al 1987) have clarified the relationship between thrombus occurrence and the site and size of myocardial infarction. Patients with anterior infarcts who have a larger extent of necrotic myocardium, particularly with aneurysms, are at a higher risk of thrombus formation than those with infarction in other areas or with a lesser degree of asynergic myocardium. The **hypothesis is that apical dyskinesia and the aneurysmic dilatation of the left ventricular apex might predispose to regional stasis of blood and consequently to thrombus development** *(Yater et al 1951, Jordan et al 1952, Asinger et al 1981b, Visser et al 1983b).*

Two-dimensional echocardiography can demonstrate the presence of unusual patterns of intra-cavitary echoes attributable to blood stasis, detectable in areas of severe apical dysfunction and predictive of thrombus development. These patterns appear as spontaneous ‘smoke-like’ echoes without defined margins, with rapid changes in configuration and acoustic intensity over a short time. More recently, the employment of qualitative and quantitative analyses with Doppler techniques has further addressed the relationship between

flow abnormalities, attributable to blood stasis, and a higher occurrence of thrombus (Delemarre et al 1990).

Therefore, the Echo-Cardiographic analysis of left ventricular wall motion abnormalities and of blood flow characteristics can allow the identification of those patients who are at higher risk of developing thrombus after acute myocardial infarction.

Incidence And Time Of Appearance Of Thrombus

The feasibility and repeatability of cardiac ultrasound, together with its high diagnostic accuracy, make this technique an ideal tool for determining the incidence and the time of appearance of left ventricular thrombi after acute myocardial infarction. The overall occurrence of left ventricular thrombus after an isolated inferior myocardial infarction has been estimated as less than 1% of the studied patients (Asinger et al 1981a, Visser et al 1983b, Weinreich et al 1984, Keren et al 1990) and is found only where aneurysmatic dilatation of the inferior wall occurs (Straton et al 1982, Wenreich et al 1984). Therefore, the clinical relevance of left ventricular thrombus in this condition appears negligible. On the other hand, the incidence of

thrombi after anterior myocardial infarction ranges from approximately 30% to nearly 60% of the patients studied (Asinger et al 1981a, Visser et al 1983b, Davis & Ireland 1986, Domenicucci et al 1990). Some series have shown a favorable effect of both anticoagulant (Norderhaug et al 1985) and fibrinolytic treatment. (Lupi et al.) in reducing the rate of thrombus formation. In a study by Vecchio et al in 1991, no difference in thrombus occurrence was observed in thrombolysed patients assigned, in a random mode, either to initial streptokinase or rt-PA therapy followed or not by subcutaneous heparin.

As far as the time of appearance is concerned, nearly 50% of thrombi develop within the first 2 days after acute infarction, with almost 95% being present within 2 weeks. (Davis & Ireland 1986, Funke Kupper et al 1989, Domenicucci et al 1990). The late development of thrombus, at more than 1 month after infarction, has been reported in approximately 10% of cases (Visser et al 1983b, Domenicucci et al 1990, Keren et al 1990). Spirito et al (1985) found a relationship between the early development of thrombus (i.e. within 48 hours of anterior infarction) and a higher global mortality; more recent

evaluations of larger patient populations have further confirmed that early thrombus formation is a marker of higher mortality, using either-in-hospital death (Domenicucci et al 1990) or global mortality rates (Funke Kupper et al 1989) as end-points. Consequently, after acute anterior myocardial infarction, an early echocardiographic search for left ventricular thrombus, performed within 48 hours of symptom onset, may be useful in attempting to identify a subgroup of patients with a poorer prognosis.

Morphological Patterns

2D – dimensional echocardiography is the best method for non–invasively assessing the anatomical characteristics of thrombi and to follow their possible variations with time.

With regard to *shape*, left ventricular thrombi can be subdivided into mural or protruding. **Mural thrombi** *have a flat configuration, with the free margin usually showing a concave curvature, parallel to the adjacent endocardial surface.* A ***thrombus is considered protruding*** *when it predominantly projects into the left ventricular cavity and the thrombus –blood interface demonstrates a curvature that is convex to*

the adjacent endocardium. **Detection of mobility** is based on the *observation of motion of a portion of the thrombus which is independent of that demonstrated by the adjacent myocardium*.

The clinical relevance of thrombus anatomy has been emphasized in recent studies which have shown a higher embolic potential for protruding and / or mobile thrombi when compared with mural, non – mobile thrombi (Haugland et al 1984, Visser et al 1985, Johannessen et al 1988

Spontaneous variations in thrombus shape, from protruding to mural configuration or vice versa, can be observed in approximately 40% of thrombi developed after anterior myocardial infarction (Domenicucci et al 1987). Changes in thrombus mobility are also frequently noted (Domenicucci et al 1987, Funke Kupper et al 1989). The majority of changes in thrombus anatomy occur in the initial time period after acute infarction: in our experience, nearly 90% of the variations in shape and mobility were detected within 1 month after infarction.

As far as the relationship between thrombus age and its morphological characteristics is concerned, patterns of protruding shape are mostly detected in the early phase after acute infarction, while in older thrombi a mural configuration appears to be predominant (Domenicucci et al 1987, Keren et al 1990).

Thrombus mobility is detectable in approximately one third of thrombi (Domenicucci et al 1987) and is usually observed only in the early period after infarction : nearly 90% of the mobile thrombi noted in the first few days after an acute infarction showed no mobility at 1 month of follow up, while all mobility was completely absent in the Echo Cardiographic examination performed at more than 6 months after infarction. Due to its peculiar transience and spontaneous variability, a reliable assessment of the incidence and evolution of thrombus mobility needs serial echocardiographic examinations repeated at short time intervals. Conceivably, the more frequent observation of mobility and the protruding configuration of left ventricular thrombus in the first few months after acute infarction is related to the occurrence of most embolic complications in the same

period. For this reason, an accurate and serial echocardiographic evaluation of thrombus anatomy in this period is advisable.

Dimension

Echocardiography permits an immediate, qualitative assessment of thrombus dimension and extent within the left ventricular cavity. Moreover, planometry of the thrombus may easily be obtained by the direct digitization of the frozen frame on the screen. However, the reliability of such a quantitative evaluation is limited by the inherent three dimensional conformation of thrombus. Serial evaluation of thrombus dimension with cardiac ultrasound has been proposed as a useful and repeatable method for assessing the effects of long-term antithrombotic treatment (Stratton & Ritchie 1984). In particular the detection of a **reduction in the maximal thickness of thrombus of over 5mm or complete thrombus resolution has been considered as attributable to the on-going antithrombotic therapy.** However, it must be pointed out that reduction in thrombus size or even its resolution may occur spontaneously in a significant percentage of patients (Spirito et al 1885, Keren et al 1990). Therefore, the presence of such spontaneous variations in thrombus dimension must be taken

into account when cardiac ultrasound is used for assessing the efficacy of chronic antithrombotic treatment.

Conversely, echocardiography is a unique tool for evaluating the acute, sometimes dramatic, effects of fibrinolytic treatment given to resolve a thrombus with the morphological characteristics consistent with high embolic potential, such as protrusion and free mobility. The complete resolution of the thrombus or a significant reduction in its size has been reported in majority of cases (Kremer et al 1985, Keral et al 1990). Other than documenting the lysis of thrombus, echocardiography can permit the visualization in real time of on-going thromboembolization with thrombus fragmentation and the subsequent appearance of thrombus fragments in the left ventricular outflow tract. Clinical evidence of thromboembolization attributable to thrombolytic treatment has been reported (Keren et al 1990) and it has been suggested that the risk/benefit ratio of this procedure still remains to be determined.

ACOUSTIC CHARACTERIZATION OF THROMBUS

QUALITATIVE AND QUANTITATIVE ANALYSIS

Cardiac ultrasound can allow both a qualitative and quantitative characterization of a left ventricular thrombus. Usually the echodensity of a thrombus is greater that that of the adjacent myocardium. The observation of an acoustically distinct structure in an area of myocardial aysnergy may itself represent a diagnostic criterion for discriminating a thin but otherwise hardly recognizable thrombus from the adjacent endocardium. Variations in reflective intensity within a thrombus may indicate differences in its histological composition or architecture (Mikell et al 1982a). Thrombi may appear as layered structures showing variations in their ehcodensity usually the most echo dense layer corresponds to the thrombus-blood interface and conceivably represents the last-formed portion of thrombus. This acoustic feature is commonly seen in fresh thrombi with a mural configuration. On the other hand, the opposite pattern may be detected in the presence of a freely mobile, swirling portion of a protruding thrombus, which demonstrates a lower echodensity and less distinct intra-cavitary margins.

Serial echocardiograms performed in the same patient occasionally demonstrate temporal variation in the acoustic characteristics of a thrombus. These may occur spontaneously or may be due to antithrombotic treatment. The spontaneous liquification of a thrombus is characterized by the presence of an echo-free space within the thrombosis substance, followed by a subsequent return to a more homogneous echodensity. Similar variations in the acoustic patterns are also detectable during acute treatment with fibrinolysis.

More recently, a technique for assessing the two-dimensional spatial distribution of echo amplitudes in a region of interest, known as quantitative texture analysis, has been employed to characterize the acoustic properties of intracardiac thrombi. Preliminary data from these studies would seem to indicate that assessment of tissue characteristics may distinguish fresh from old thrombi and help to identify those thrombi that are at a higher risk of systemic embolization. (Lloret et al 1985, Bellotti et al 1991). If these results are confirmed, they open a new possible application of cardiac ultrasound in the definition of the embolic potential of left ventricular thrombi.

PREVENTION OF EMBOLIC COMPLICATIONS

Contribution Of Cardiac Ultrasound

. The systematic prevention and treatment of left ventricular thrombus with anticoagulants still appears questionable. First such treatment does not totally prevent thrombus development, and late thrombus formation or recurrence after discontinuation of the drug has been reported. (Asinger et al 1981a, Visser et al 1983a, Keren et al 1990). Secondly, the potential benefits of this treatment must be weighed against the hazards of possible haemorrhagic complications. For these reason, it would be of benefit if echocardiography could identify those patients at higher risk of embolization in order to select out the subgroup of patients who might benefit from antithrombotic treatment, thus minimizing the danger of haemorrhagic complications. Although the anatomical instability of thrombi may limit the value of previous echo cardiographic studies performed in order to define the morphological markers of higher embolic potential, from the analysis of the data available so far, it is reasonable to state that a protruding pedunculated thrombus with a freely mobile portion represents a very likely source of embolization and would constitute an indication for anti-thrombotic treatment, if no major contraindications are present. As

far as the effects of such treatment are concerned, cardiac ultrasound can be employed to verify the possible success of anticoagulant therapy, considered as either resolution of thrombus or induced favorable changes of its morphology into a mural, non-mobile configuration. This finding can be a reason for stopping the treatment, however; further echocardiography control is advisable in order to detect the potential recurrence of thrombi with high embolic risk.

Best Timing For Echocardiographic Evaluation Of Left Ventricular Thrombus.

Due to the anatomical instability of left ventricular thrombus, an accurate assessment of its natural history with cardiac ultrasound needs repeated examinations performed at short time intervals, particularly in the first month after infarction, when most morphological variations occur. However, as far as the clinical definition of thrombus is concerned, a limited number of echocardiograms appear suitable to identify the most crucial events during its dynamic evolution.

After anterior myocardial infarction, an **early examination should be performed within 24-48 hours of symptom onset,** this

would allow the detection of the abnormalities in left ventricular wall motion and flow patterns, which identify a subgroup of patients more prone to thrombus formation. In addition, the observation of early thrombus development may help in discriminating patients with a poorer clinical profile and a higher risk of death.

A **second echocardiogram should be obtained 10 to 15 days after infarction or at the time of hospital discharge**. This evaluation would allow the detection of thrombi of high embolic potential.

Finally, a **third echocardiogram is advisable 1- 3 months after the infarction.**

ECHOCARDIOGRAM

ECHOCARDIOGRAM

Patients were serially evaluated by 2D and Doppler Echocardiography in the following sequence: within 48hrs, day 14 and at 1month after infarction. All examinations were performed using a **ALOKA SSD 2000** machine 2.5Mhz transducer and the were stored on VHS tapes for later analysis.

The diagnosis of Left Ventricular Thrombosis was made when an echodense mass with a margin distinct from the left ventricular wall was detected within the left ventricular cavity and was visible throughout the cardiac cycle in atleast two different echocardiographic views and associated with asynergy (akinesis or dyskinesis) of the adjacent myocardium.

During recording, particular care was taken to obtain all possible views and to optimize depth and gain settings in order to minimize the possibility of false positive or false negative readings. B-colour was also used in equivocal situation. Each echocardiographic study was

interpreted independently by two echocardiographers who were blinded to patients' clinical data.

Left ventricular end–diastolic and end–systolic volumes and ejection fraction were determined from apical two-and four chamber views using the Simpson's biplane formula, according to the recommendation of the American Society of Echocardiography. Tracing of endocardial borders in end diastole and end systole were performed on the ALOKA machine in the technically best cardiac cycle. The volumes were normalized for Body surface area and expressed as indexes.

In order to calculate the wall motion score index, the left ventricle was divided into 17 segments. Segmental wall motion was graded as follows: normal motion at rest (Score1), hypokinetic – marked reduction in endocardial motion and systolic thickening (Score – 2), akinetic – virtual absence of inward motion and systolic thickening (Score – 3) and dyskinetic – paradoxical wall motion away from the centre of the left ventricle in systole (Score – 4). The Wall

motion score index was calculated by summation of individual segment scores divided by the number of interpreted segments.

Pulsed-wave and color Doppler flow mapping were used for the detection and grading of mitral regurgitation. Severity of mitral regurgitation was assessed semi quantitatively according to the length of the turbulent flow jet into the left atrial cavity.

Myocardial necrosis, once established, results in a permanent WMA. The degree of transmural involvement required before wall motion becomes abnormal is approximately 20%. The implication of this is that non-transmural MI will result in hypokinesis or akinesis of the wall, even though the majority of the myocardial mass may still be perfused and viable. (Braunwald 7^{th} Edn. Pg. 242-245)

In clinical Echo, a RWMA, conforming to a known coronary distribution is the hall mark of acute ischemia or MI. After successful reperfusion, WMA typically will resolve. The time course over which they resolve is variable and may range from 12 hours to 2 weeks.

Typically, WMA recover within 72 hours if blood flow has been restored in a timely fashion. (Braunwald 7th Edn. Pg. 243)

Areas of abnormal regional wall motion are observed almost universally in patients with Myocardial infarction and the degree of WMA can be categorized with a semi quantitative WMSI. Of note, abnormal wall motion is less often noted on echocardiography. When the infarction is small and the age of RWMA can't always be determined, LV function estimated from 2D echo correlates well with measurements from angiography and is useful in establishing prognosis after Myocardial infarction. Furthermore, the early use of echo can aid in the early detection of potentially viable but stunned myocardium (contractile reserve), residual provocable ischemia, patients at risk for the development of CHF after myocardial infarction, and mechanical complications of Myocardial infarction. (Braunwald 7th Edn. Pg. 1162 & 1163)

Prospective studies have suggested that patients who develop a mural thrombus early (with in 48 to 72 hours of infarction) have an extremely poor early prognosis, (Ref :- Barbera S, Hillis LD : Echo

cardiographic recognition of LV mural thrombus Echocardiography 16 : 289, 1999). with a high rate of mortality from the complications of a large infarction(shock, reinfarction, rupture and ventricular tachyarrythmia), rather than embolism from the LV thrombus. (Braunwald 7th Edn. Pg. 1215)

Management Of LV Thrombus

I.V. Heparin (apTT 1:5-2) followed by 3-6 months Warfarin. In

(1) an embolic event has already occurred. (or)

(2) The patient has a large amount whether or not a thrombus is visualized echocardia graphically.

(3) Other than the AWMI if a thrombus or large WMA is detected.

Aspirin, although probably not capable of affecting thrombus size in most patients, may prevent further platelet deposition on existing thrombi and also is protective against recurrent ischemic events. (Braunwald 7th Edn. Pg. 1215)

AIMS OF THE STUDY

AIMS OF THE STUDY

- ★ To study the incidence / prevalence of intra-cavitary thrombus in STEMI patients following thrombolysis.

- ★ To establish the role of Early Echo Cardiogram in studying the relationship between wall motion abnormality and thrombus formation.

- ★ To evaluate associated Co-morbid condition (Hypertension, Diabetes Mellitus, Smoking, Age and Sex, Area of infarct etc) in relation to intra-cavitary thrombus formation in post – thrombolytic STEMI patients.

MATERIALS AND METHODS

MATERIALS AND METHODS

Materials

Settings	**:**	Intensive Coronary Care Unit (ICCU) Government Rajaji Hospital Madurai
Type of Study	**:**	Prospective Cohort study
Period of Study	**:**	August 2005 – July 2006
Sample Size	**:**	144 patients
Consent	**:**	Informed consent was obtained from all Patients or relatives

Inclusion Criteria

All patients admitted to ICCU with STEMI

STEMI irrespective of the type, age and the extent of successful thrombolysis were included in this study.

Exclusion Criteria

- ★ Previous H/O thrombolysis
- ★ Previous H/O intracavitary thrombus
- ★ Arrythmias
- ★ Myocarditis
- ★ LBBB
- ★ Old MI
- ★ Acute MI with Median delay > 24 hours

Laboratory Materials

The following investigations were done for all the patients.

- ★ Hb, TC, DC, ESR
- ★ Blood sugar, urea, creatinine, serum electrolytes, lipid profile, ECG
- ★ Enzyme markers CKMB – TOTAL

  Baseline Echo was estimated on Day 1, Day 14 and then on Day 28.

Patients were monitored and managed in ICCU as per the standard guidelines. Appropriate biochemical tests were done according to their clinical condition and underlying medical diseases.

METHODS

We have prospectively evaluated 144 consecutive patients with first acute myocardial infarction who met the following criteria: all cases of STEMI irrespective of the type of infarction, age or sex of the patient.

Baseline characteristics of patients enrolled in the study are as follows:age,sex,risk factors(smoking, SHT,DM,Dyslipidemia,Family H/o CAD,Menopause),median delay less than 24hrs,presence of MR and whether or not patient underwent thrombolysis.

Clinical assessment of patient's cardiovascular status was made according to the Killip classification.

Therapy

Thrombolytic therapy (iv streptokinase 1.5million IU over 30 to 60 minutes) was administered in 110/144 (76.4%) patients. The remaining patients did not receive thrombolytic therapy due to some contraindications.

Definitions

1. **STEM 1** → ST Elevation
    a. 1 mm in Limb leads
    b. 2 mm in precordial leads

2. **Successful thrombolysis** → Resolution of chest pain x > 50% reduction of ST elevation

3. **Unsuccessful thrombolysis** → Non resolution of chest pain and < 50% reduction of ST elevation

4. **Median Delay** → Time interval between onset of chest pain to admission in ICCU.

5. Killip Classification

Class I:No signs of pulmonary and venous congestion

Class II: Moderate heart failure as evidenced by rales at the lung bases,S3 gallop,tachypnea or signs of right heart failure including venous and hepatic congestion

Class III: Severe heart failure, pulmonary edema

Class IV:Shock with systolic pressure<90mmHg and evidence of peripheral cyanosis, mental confusion and oliguria.

6. **Thrombolytic Agent and dose** → Streptokinase dose I.V. 1.5 million units in 100 ml of normal saline within 1 hour.

7. **Antithrombotic effect**
    a. Heparin
    b. Clopidogrel
    c. Warfarin

8. **Higher Median Delay:** Median delay>12hrs but < 24hrs.

Clinical and biochemical data were collected using a Proforma. All the relevant details were fed into a computer**.** End point of the study was discharge from ICCU.

Computer Analysis of data was done using the software epidemiological information package – 2002 developed by centers for disease control and prevention, Atlanta in collaboration with WHO.

Chi – square test was used for tests of significance.

ANALYSIS AND RESULTS

ANALYSIS AND RESULTS

144 patients of Acute Coronary Syndrome admitted in ICCU, Government Rajaji Hospital, Madurai were included in the study.

CHARACTERISTICS OF STUDY CASES

Table 1

Age distribution of the Study Group

Age Group	Patients	
	No.	**%**
21-30	3	2.1
31-40	17	11.8
41-50	41	28.5
51-60	46	31.9
> 60	37	25.7
Total	144	100
Mean	57.6	
S.D.	12	

Majority of patients in the study group belonged to 4^{th} and 5^{th} decade.

Table 2

Sex	Patients	
	No.	**%**
Male	134	93.1
Female	10	6.9

Male formed the majority of patients admitted in ICCU (93.1%). Most of them belong to 4^{th} and 6^{th} decade.

Table 3

Risk factors

Risk Factors	Present		Absent	
	No.	**%**	**No.**	**%**
Smoking(134) among men	77	57.5	57	42.5
SHT	51	35.4	93	64.6
DM	30	20.8	114	79.2
Dyslipidemia	105	72.9	39	27.1
CAD	16	11.1	128	88.9
Menopause	9	90	1	10

Smoking (in men), dyslipidemia and systemic hypertension were the major risk factors of patients in the study group.

Table 4

No. of Risk factors present in patients

No. of risk factors present	Patients	
	No.	%
Nil	9	6.3
1	45	31.5
2	46	36.2
3	31	21.7
4	9	6.3
5	3	2.1
6	-	-

Number of patient with 1 or 2 risk factors formed 33.81%.

Table 5

Area of Infarct

Area of infarct	Patients	
	No.	**%**
AWMI	95	66
Others	39	27.1
Both	10	6.9

AWMI accounted for the majority (66%) of patients in study group.

Table 6

Thrombolysis

S.No.	Thrombolysis	Patients	
		No.	%
1.	Successful thrombolysis	76	52.8
	Unsuccessful thrombolysis	34	23.6
	Total	110	76.4
2.	Non Thrombolysis	34	23.6

Two thirds in the study group underwent thrombolysis of which two thirds had successful thrombolysis defined as "50% ST segment resolution and or resolution of chest pain".

CHARACTERISTICS OF PATIENTS AND INCIDENCE OF CLOT

Table 7

Age and Incidence of Clot

Age Group	Total Cases	LV Clot			
		Present		Absent	
		No.	%	No.	%
21-30	3	1	33.3	2	66.7
31-40	17	8	47.1	9	52.9
41-50	41	11	26.8	30	73.2
51-60	46	9	19.6	37	80.4
> 60	37	7	18.9	30	81.1
Total	144	36	25	108	75
Mean S.D.		53.61 11.25		57.78 10.08	
P		0.0448			

Statistically significant relationship exists between age and incidence of LV Clot.

Table 8

Sex and Incidence of Clot

Sex	LV Clot			
	Present		Absent	
	No.	%	No.	%
Male	34	94.4	100	92.6
Female	2	5.6	8	7.4
p	0.5229			

No statistically significant relationship exists between sex and incidence of clot.

Table 9

Risk Factors and Incidence of Clot

Risk Factors	LV Clot			
	Present (36)		Absent (108)	
	No.	%	No.	%
Smoking				
Yes	22	61.1	55	50.9
No	14	38.9	53	49.1
p	0.3853			
SHT				
Yes	15	41.7	36	33.3
No	21	58.3	72	66.7
p	0.4813			
DM				
Yes	7	19.4	23	21.3
No	29	80.6	85	78.7
p	0.8133			
Dyslipidemia				
Yes	26	72.2	79	73.1
No	10	27.8	29	26.9
p	0.9137			
CAD				
Yes	2	5.6	14	13
No	34	94.4	84	87
P	0.1812			

Table 10

Risk Factors	LV Clot				p
	Present (36)		Absent (108)		
	No.	%	No.	%	
Menopause					
Yes	2	5.6	7	6.5	
No	34	94.4	101	93.5	0.6007
No. of Risk factors					
Nil	2	5.7	7	6.5	0.6007(N.S.)
1	10	28.6	35	32.4	0.7555(N.S.)
2	10	28.6	36	33.3	0.6798(N.S.)
3	11	31.4	20	18.5	0.1979(N.S.)
4	2	5.7	7	6.5	0.6007(N.S.)
5	-	-	3	2.8	0.4189(N.S.)

*NS – Not Significant

No statistically significant relationships exist between individual risk factors and LV Clot occurrence.

Table 11

KILLIP Classification and Incidence of Clot

Killip Classification	LV Clot			
	Present		Absent	
	No.	%	No.	%
Nil	2	5.6	14	13
I	8	22.2	61	56.5
II	22	61.1	27	25
III	1	2.8	2	1.9
IV	3	8.3	4	3.7

Incidence of LV Clot more in those under Killip Class II.

Table 12

Area of Infarct and Incidence of Clot

Area of Infarct	LV Clot			
	Present		Absent	
	No.	%	No.	%
AWMI (95)	31	32.6	64	67.4
IWMI(39)	1	2.7	38	97.3
Both (10)	4	40	6	60
P	0.0061			

Area of infarct and incidence of clot have significant relationship.

Table 13

Thrombolysis and Incidence of Clot

Thrombolysis	LV Clot			
	Present		Absent	
	No.	%	No.	%
Successful thrombolysis	16	44.4	60	55.6
Unsuccessful thrombolysis	8	22.2	26	24.1
Total	24	66.6	86	79.7
Non Thrombolysis	12	33.3	22	20.3
Total	36	100	108	100
P	0.174 (Not significant)			

Incidence of thrombus in thrombolysed individual was 44.4% and 33.3% in non thrombolysed patients which was not statistically significant.

Table 14

Presence of MR and Incidence of Clot

Presence of MR	LV Clot			
	Present		Absent	
	No.	%	No.	%
Present (38)	15	39.5	23	60.5
Absent (106)	21	19.8	85	80.2
P	0.029(Significant)			

The presence of MR on serial echo was found to have statistically significant relationship with development of LV clot.

Table 15

Median Delay and Incidence of Clot

Median Delay	LV Clot			
	Present		Absent	
	No.	%	No.	%
< 6 hours	13	36.1	60	55.6
6.1-12	12	33.3	43	43
12.1-24	2	5.6	1	0.9
> 24	9	25	4	3.7
Mean delay in hours S.D.	12.06 9.87		6.95 4.34	
P	0.0059			

Patients with LV clot have higher median delay than patients without LV clot and this difference is statically significant.

Table 16

Echo evaluation parameter and Incidence of Clot

LV Clot	LVIDd		LVEF		EDV		ESV		FS		WMSI	
	Mean	**S.D.**	**Mean**	**S.D.**	**Mean**	**S.D.**	**Mean**	**S.D.**	**Mean**	**S.D.**	**Mean**	**S.D.**
Present	5.02	0.36	40.33	4.95	140.14	29.68	48.19	6.15	57.13	14.83	1.52	0.18
Absent	4.89	0.91	48.28	9.91	129	28.9	26.44	5.96	63.23	9.74	1.25	0.12
P	0.1268 (Not significant)		0.0001 (Significant)		0.2582 (Not Significant)		0.0001 (Significant)		0.5213 (Not Significant)		0.0001 (Significant)	

Statistically significant relationships exist between occurrences of LV Clot and ESV, WMSI, LVEF.

Table No. 17

Days from acute myocardial infarction and

percentage of thrombosis

Days	Thrombolysis	
	No.	%
1-7	17	47.2
8-14	11	30.6
15-28	8	22.2
Total	36	100

It was observed that most (47.2%) of the patients developed left ventricular thrombus within the first week of post infarct period.

DISCUSSION

144 Patients admitted to ICCU with acute myocardial infarction, in Government Rajaji Hospital, Madurai during the period of August 2005 to July 2006 were included in the study. End point of the study was discharge from the ICCU one month after infarction.

Age of the patients admitted to ICCU ranged from 21 – 80 years with a mean age of 57.6±12. 93.1% of the patients admitted to intensive coronary care unit were males.

Serial Echo's when done at the time of admission and on Days 14 and 28 to assess whether Echo cardiographic scores (data) are useful as predictors of LV thrombus occurrence.

25%(36) of the patients developed LV thrombosis during the study. Of the 36 patients who developed LV thrombosis,16 patients (15%) of the thrombolysed group and 10 patients (29%) of the non thrombolsyed group had WMSI > 1.5. Of the remaining 10 patients who developed thrombus, 6 (5%) patients were thrombolysed and 4

(12%) patients were non thrombolysed, and their WMSI score was 1-1.5.This indicates that the apparent decreased incidence of LV thrombus formation in thrombolysed patient is not due to thrombolysis *per se*, rather clearly depicts the impact of WMSI on development of LV thrombus.

Patients with WMSI > 1 were found to have increased chance of development of LV thrombus, the risk being substantially increased for score > 1.5.

36 patients who developed LV thrombus (both thrombolysed and non thrombolysed group) had high end systolic volume on base line Echo. Hence high end systolic volume in acute myocardial infarction is also an independent predictor of left ventricular thrombus.

The incidence of left ventricular thrombi in our study was 25% which is lower than previously published data **European Heart Journal (1993) 19, 908-916 A.D. Popovic et al.** This is probably result of absence of long term follow up protocols in our study.

36 patients who developed LV thrombus 31 (32.6%) had AWMI which agrees with earlier studies that document the rarity of LV thrombus in inferior wall infarct **(Lea and Febiger European Heart Journal 1994; 575-625).**

SUMMARY

The "STUDY OF INTRACAVITARY THROMBUS IN THROMBOLYTIC ERA" was conducted among 144 patients admitted in ICCU, Govt.Rajaji Hospital, Madurai.

From the patients who satisfied inclusion criteria, serial echo's were done at the time of admission(baseline) and on day 14 and day 28 after infarction to detect the occurrence of LV thrombus. The relationship between WMSI (Wall motion score index),site of infarct, elevated ESV and formation of thrombus was evaluated.

Significant statistical association was noted with respect to the following factors with incidence of LV thrombus as shown below:

- ★ 47.1% in 4th decade,
- ★ 33.3%in 3rd decade,
- ★ Anterior wall MI: 32.6%,
- ★ 39.5% in the presence of MR,
- ★ High initial ESV at baseline echo and
- ★ WMSI >1.5

No statistically significant relationship was noted with relation to sex, smoking, presence of systemic hypertension, diabetes mellitus and dyslipidemia,menopause and family h/o CAD and formation of LV Thrombus.

The study clearly indicates that in patients with AMI, high initial ESV at baseline echo and WSMI>1.5 are independent predictors of later development of LV thrombus.

CONCLUSION

- The incidence of intra cavitary thrombus in STEMI patients following thrombolysis was 21.81%.

- Identification of high WMSI on admission (base line echo cardiogram) plays a definite role in predicting development of LV Thrombus.

- The analysis revealed age, sex, smoking, systemic hypertension, diabetes mellitus, dyslipidemia, family history, CAD were not independent predictors of LV thrombus formation.

- LV Thrombosis was associated with higher initial end systolic volume index, lower initial LV ejection fraction, killip class > 1, higher median delay, occluded infarct related artery, and non thrombolytic therapy.

- It appears that determination of WMSI and end systolic volume can differentiate patient with acute STEM1 who would develop LV thrombosis from those who would not.

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PROFORMA

STUDY OF INTRA CAVITARY THROMBUS IN THROMBOLYTIC ERA

Name : Age : Sex : C.D. No.:

Address : I.P.No. :

DOA :

DOE :

DOHS :

☐ DM ☐ HT

☐ SMOKING ☐ CAD

HLP TC TG HDL-C VLDL-C LDL-C

☐ MENOPAUSE ☐ EMBOLISM

☐ OTHERS

IN HOSPITAL

☐ CVA ☐ DVT ☐

HYPOTENSION

☐ Killip Classification I II III IV ☐ LVF

ECG

RHYTHM ☐ Sinus

AF

AREA OF INFARCT

Median Delay

Success Yes/No

50% ST resolution

Resolution of Chest pain

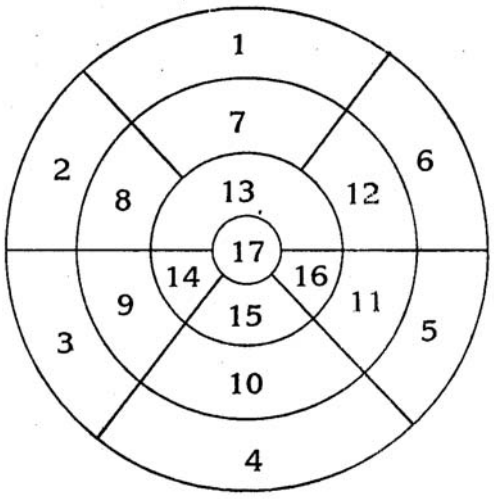
ECHO EVALUATION

IVSd IVSs LA

LVIDd LVIDs AO

LVPWd	LVPWs	RA
EF	FS	RV
ESV	EDV	SV
MR		
Clot : Location	Size	Mobility
Other		

New Segment No. & Score		
01.	Basal Anterior	
02.	Basal Anterior septal	
03.	Basal Inferior septal	
04.	Basal inferior	
05.	Basal Inferior Lateral	
06.	Basal Anterior Lateral	
07.	Mid Anterior	
08.	Mid Anterior Septal	
09.	Mid Inferior Septal	
10.	Mid Inferior	
11.	Mid Inferior Lateral	
12.	Mid Anterior Lateral	
13.	Apical Anterior	
14.	Apical Septal	
15.	Apical Inferior	
16.	Apical Lateral	
17.	True Apex	
	Total	

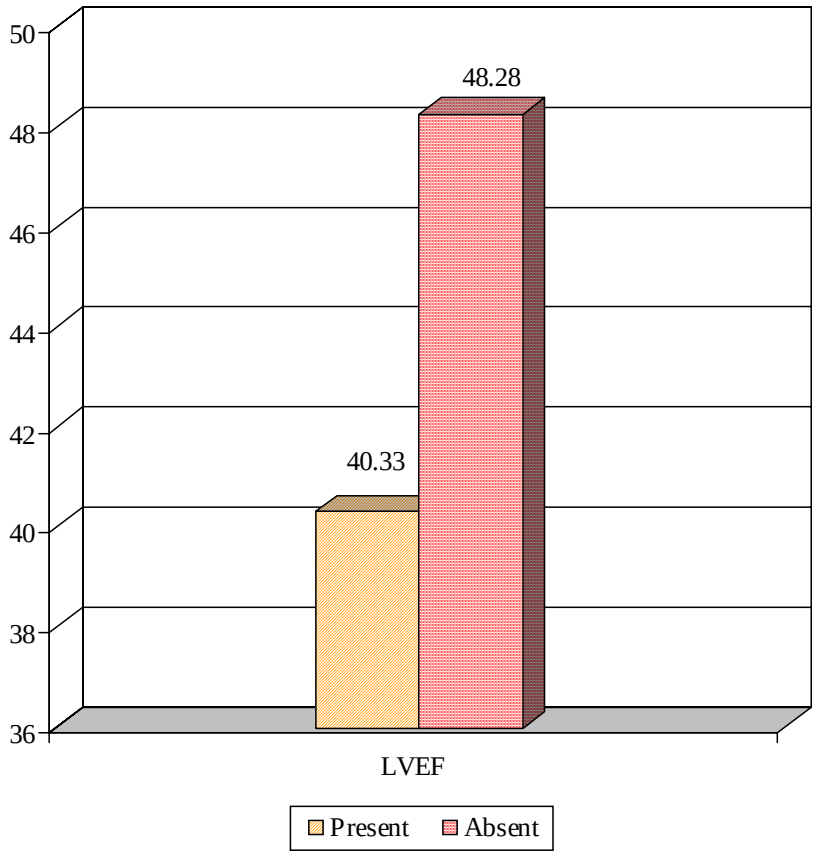


WMSI (Wall Motion Score Index) = Total Score / No. of segments evaluated

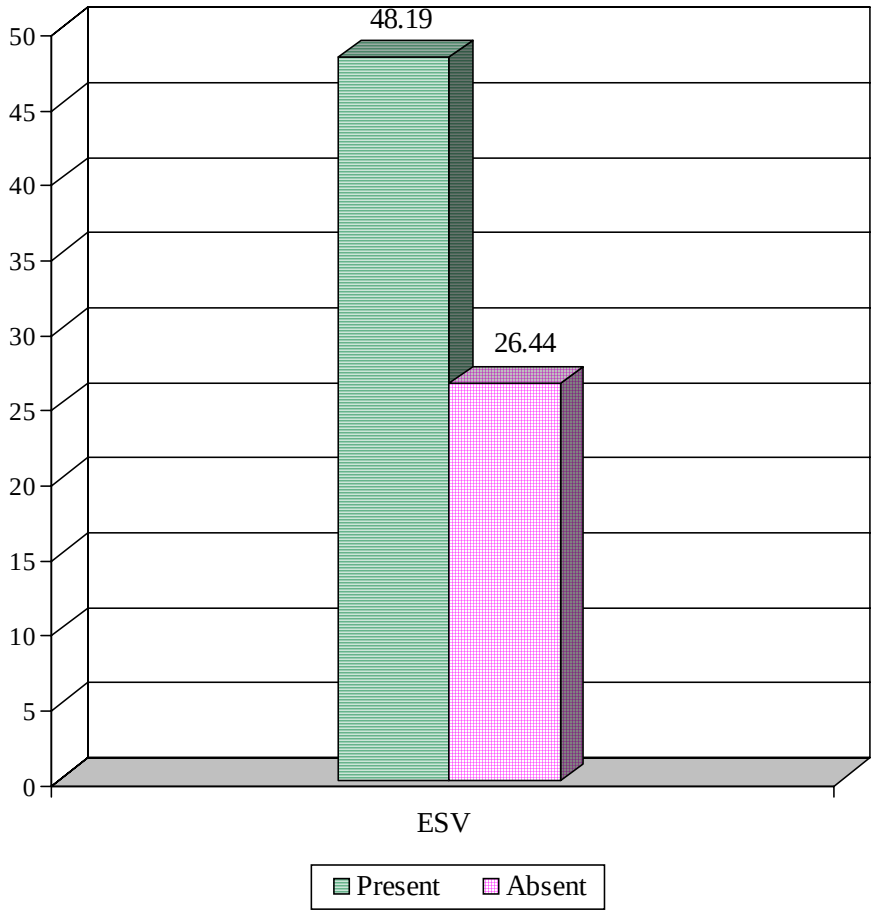
☐ NORMAL – 1 ☐ HYPOKINECTIC – 2 ☐ AKINETIC – 3

☐ DYSKINETIC-4 ☐ ANEURYSM-5

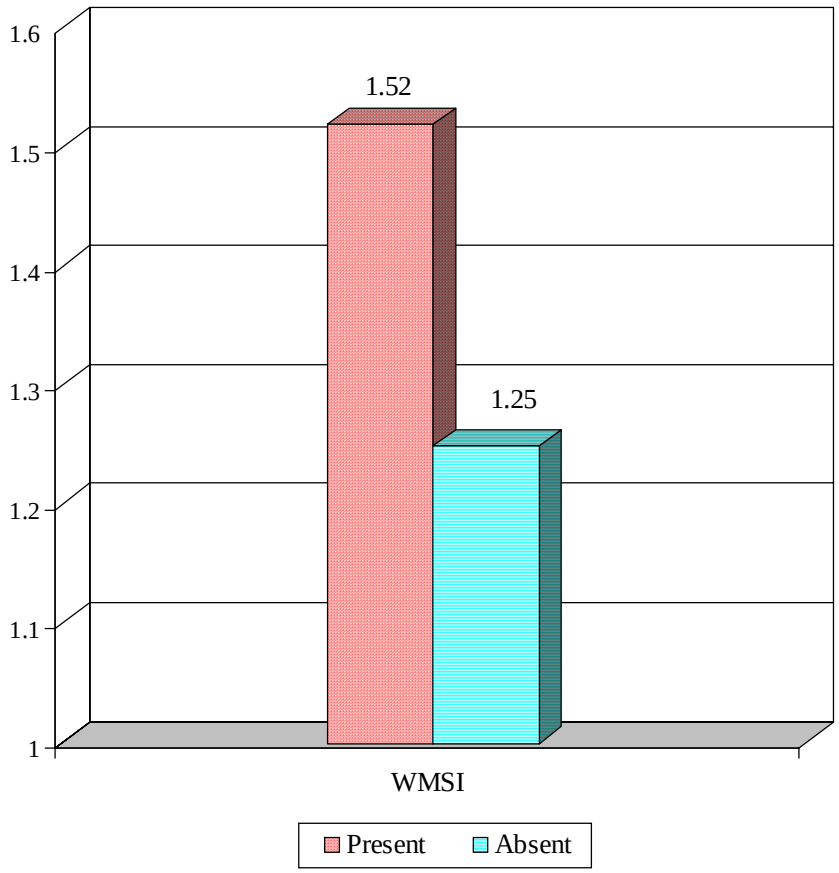
LVEF and Incidence of LV Clot



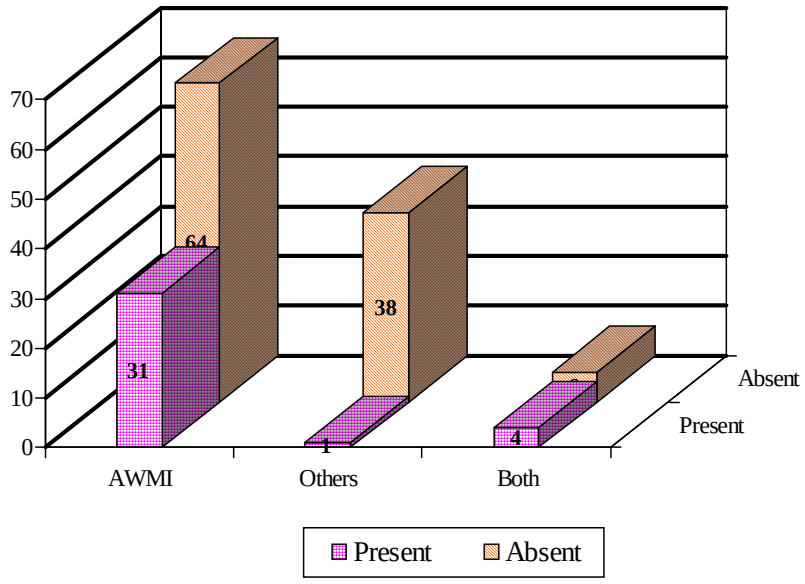
ESV and Incidence of LV Clot



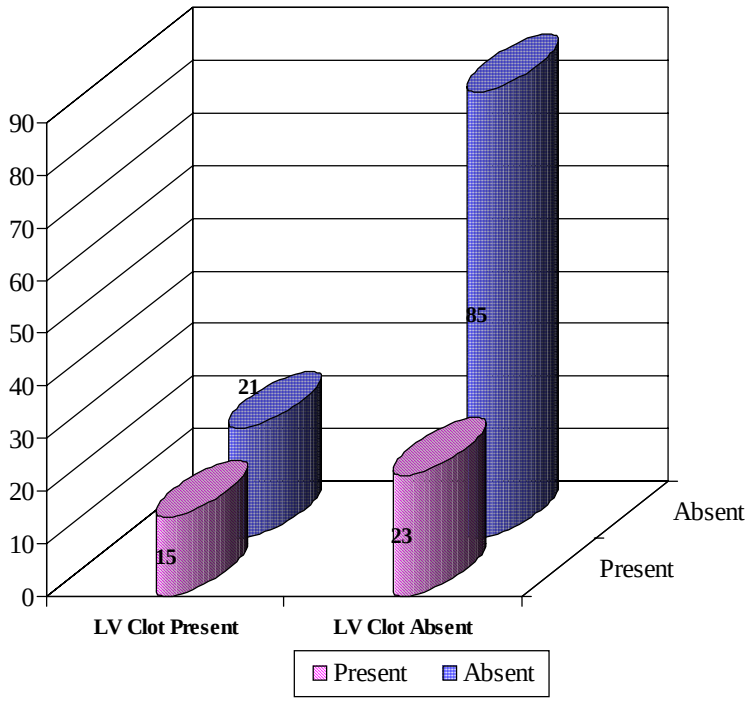
WMSI and Incidence of LV Clot



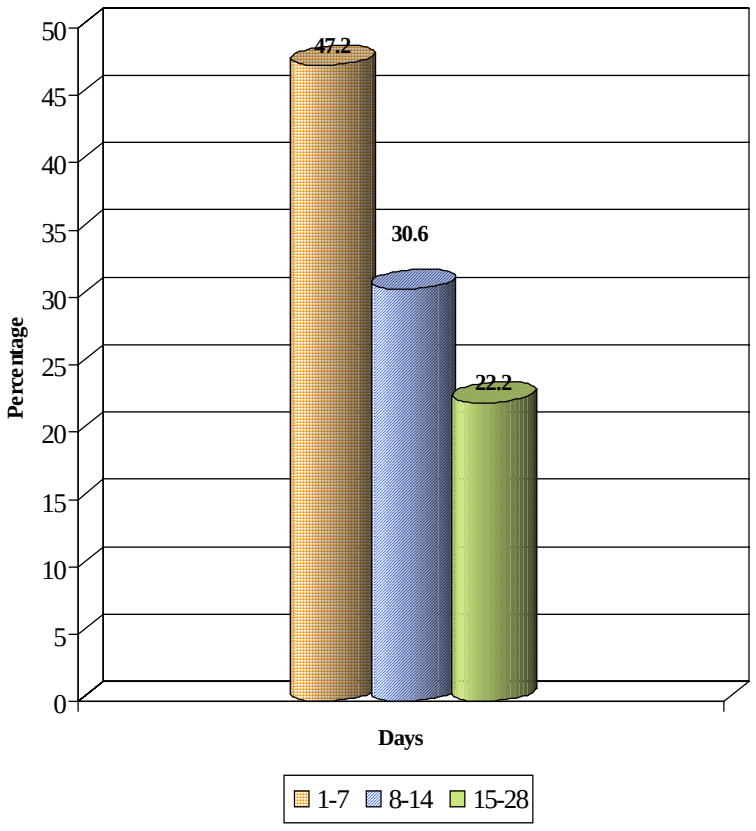
Area of Infarct and Incidence of Clot



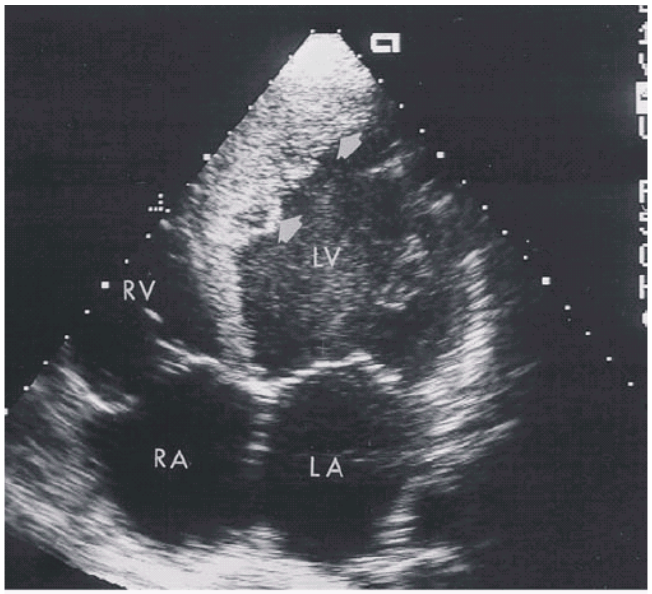
Presence of MR and Incidence of Clot



Days from acute myocardial infarction and percentage of thrombosis

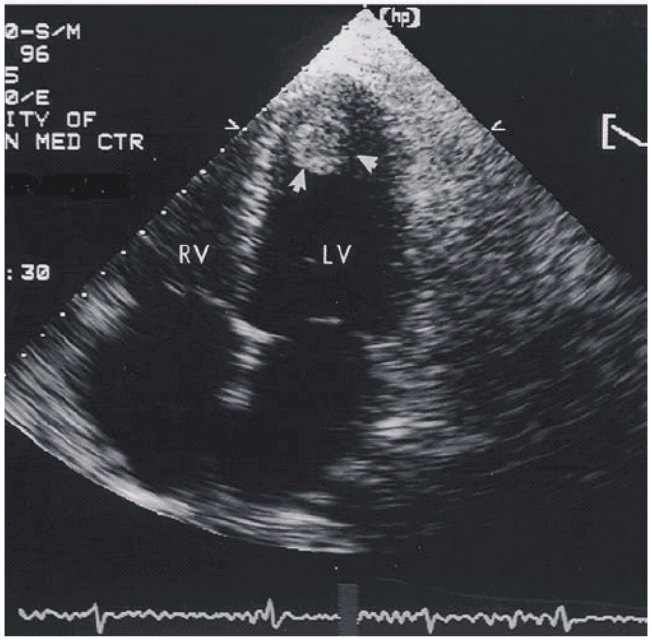


ECHO CARDIOGRAPHIC PICTURE OF LV THROMBUS



A

Apical four-chamber view reveal a large laminar thrombus filling a subsdantial portion of the apex and adherant to the ventricular septum (Arrows - top panel).



The bottom panel denotes a smaller, more spherical thrombus in the apex of the left ventricle.

MASTER CHART

S.No.	Age	Sex	Smoking	SHT	DM	HDL	LDL	TGL	CAD	Median delay in hours	Killip classification	Area of Infarct AWMI	Others	Thrombolysis successful	Unsuccessful	LVIDd	LVEF	EDV	ESV	FS	WMSI	LV clot	MR	TX/ Not Tx
1	6	M	1	1	2	1	3	3	2	4	2	1	2	1	2	5.8	46	174	93	24	1.71	1	1	
2	5	M	2	2	2	1	3	1	2	24	2	1	2	2	2	5.9	44	170	95	22	1.5	1	1	NT
3	5	M	2	2	2	1	3	3	2	16	2	1	2	2	2	5.8	47	162	86	21	1.71	1	1	NT
4	6	M	1	2	2	1	2	3	1	5	1	2	1	1	2	5	70	140	42	27	1.72	2	1	
5	5	M	1	2	2	2	3	3	2	4	1	1	2	1	2	4.9	65	142	50	22	1.24	2	2	
6	6	M	2	1	2	1	3	3	2	6	1	1	2	1	2	4.6	68	130	42	22	1.24	2	1	
7	6	M	1	2	2	1	2	2	2	24	3	1	2	2	2	5.9	45	170	92	23	1.53	1	2	NT
8	7	M	1	2	2	2	3	2	2	4.5	1	2	1	1	2	4.7	70	140	42	24	1.18	2	1	
9	5	M	2	2	2	2	3	3	2	2	2	1	2	1	2	4.9	42	137	42	21	1.24	2	2	
10	5	M	2	2	2	2	3	2	2	4.5	1	2	1	1	2	4.7	70	187	26	29	1.12	2	2	
11	7	F	2	1	1	2	3	3	1	8.5	2	2	1	2	2	5.7	45	110	60	21	1.12	2	2	
12	6	M	2	1	1	2	3	2	2	8	1	1	2	2	2	4.9	47	188	46	23	1.12	2	2	
13	4	M	1	2	2	2	3	3	2	4	2	1	2	1	2	6.3	39	102	84	22	1.82	1	1	
14	7	M	2	1	2	2	2	2	2	11.5	1	1	2	2	2	5.3	37	87	54	20	1.35	2	1	
15	4	M	1	2	2	2	3	3	2	2.5	5	1	2	1	2	5.7	33	90	35	19	1.24	2	1	
16	6	M	1	2	2	2	2	3	2	5.5	5	1	2	1	2	5.6	40	98	41	21	1.35	2	1	
17	3	M	2	2	2	2	3	2	2	7.5	2	1	2	2	1	5.8	46	106	82	24	1.24	1	2	
18	6	M	2	2	2	2	3	2	2	8	5	1	2	2	1	4.3	42	90	42	21	1.24	2	1	
19	5	M	2	1	1	2	3	3	1	5	5	2	1	1	2	5.7	47	98	58	23	1.12	2	2	
20	8	M	1	1	1	2	2	2	2	10	2	1	2	1	2	5.9	45	94	86	23	1.53	1	1	
21	6	M	1	1	1	2	3	3	1	12	5	1	2	2	2	5.2	44	96	64	23	1.24	2	1	
22	7	F	2	2	2	2	3	2	2	6	5	1	2	1	2	4.2	45	130	47	24	1.18	2	1	
23	8	F	2	2	2	1	3	3	2	48	2	1	2	2	2	5.8	47	143	78	24	1.59	1	1	NT
24	6	M	1	1	2	2	2	2	2	4	5	2	1	1	2	4.1	46	138	42	23	1.12	2	1	

S.No.	Age	Sex	Smoking	SHT	DM	HDL	LDL	TGL	CAD	Median delay in hours	Killip classification	Area of Infarct AWMI	Others	Thrombolysis successful	Unsuccessful	LVIDd	LVEF	EDV	ESV	FS	WMSI	LV clot	MR	TX/ Not Tx
25	9	F	2	2	2	2	2	2	2	3.5	5	2	1	1	2	4.7	47	87	47	24	1.24	2	1	
26	6	M	2	2	2	2	3	2	2	3.5	5	1	2	1	2	6.8	28	70	38	17	1.24	2	1	
27	7	M	2	2	2	2	2	2	2	3	5	1	2	1	2	5.9	39	80	42	19	1.29	2	1	
28	7	M	1	1	2	2	3	3	2	6	5	1	2	2	2	6	58	86	36	20	1.24	2	2	
29	5	M	1	1	1	2	2	2	1	24	4	1	1	2	2	5.8	40	176	84	21	1.82	1	2	NT
30	5	M	1	2	2	2	3	3	2	4	1	1	2	1	2	4.7	41	90	37	20	1.24	2	1	
31	6	M	1	2	1	2	2	3	2	5	1	1	2	1	2	4.6	56	136	44	28	1.24	2	1	
32	5	M	2	2	2	1	2	3	2	15	4	1	1	2	1	6.2	37	170	87	21	1.94	1	1	
33	6	M	1	2	2	2	3	2	2	4	2	1	2	1	2	4.6	40	97	38	21	1.29	2	1	AF
34	4	M	1	2	2	1	2	3	2	24	2	1	2	1	2	6.6	34	176	94	19	1.76	1	2	
35	5	M	2	2	2	2	3	2	2	2.5	1	2	1	1	2	4.1	47	92	42	23	1.12	2	1	
36	6	M	2	2	2	2	3	2	2	4	2	1	2	1	2	4.8	53	192	52	24	1.24	2	2	
37	7	M	2	1	2	2	3	3	2	24	1	1	2	2	2	4.6	46	160	46	22	1.29	2	2	
38	5	M	1	1	1	1	2	3	2	2.5	2	1	2	1	2	6.2	35	180	94	20	1.53	1	1	
39	7	M	1	1	2	2	3	2	2	6.5	2	1	2	2	1	6.3	28	184	94	18	1.71	1	1	
40	7	M	2	1	2	2	3	2	2	6.5	2	1	2	2	2	6.5	35	167	86	19	1.47	1	1	NT CVA N
41	7	M	1	1	2	2	2	3	2	11	1	2	1	2	2	4.4	28	97	34	18	1.35	2	2	
42	6	M	1	1	1	2	2	2	2	4	1	2	1	1	2	4	52	140	42	26	1.24	2	2	
43	5	M	2	1	1	2	2	3	2	3	1	1	2	1	2	4.7	35	82	43	19	1.47	2	2	
44	7	M	2	2	2	2	2	3	2	6.5	1	1	1	2	1	4.3	67	140	37	29	1.12	2	2	
45	6	M	2	1	2	2	2	2	2	6	2	1	2	1	2	5.8	47	170	87	23	1.53	1	2	
46	3	M	1	1	2	1	3	3	1	6	3	1	1	1	2	4.6	74	106	35	30	1.35	2	2	
47	7	M	1	1	1	2	3	2	2	3.5	2	1	2	1	2	5.7	49	156	64	21	1.47	2	2	
48	5	M	1	2	2	1	2	3	2	6	1	1	2	1	2	5.7	44	160	54	22	1.47	2	2	
49	6	M	1	1	1	2	3	3	2	9	1	1	2	2	1	5.6	42	160	61	21	1.59	2	2	

S.No.	Age	Sex	Smoking	SHT	DM	HDL	LDL	TGL	CAD	Median delay in hours	Killip classification	Area of Infarct AWMI	Others	Thrombolysis successful	Unsuccessful	LVIDd	LVEF	EDV	ESV	FS	WMSI	LV clot	MR	TX/ Not Tx
50	6	M	2	1	1	1	3	3	1	11	2	1	2	2	1	7.2	26	170	64	17	1.47	2	1	LVA
51	6	M	1	2	2	2	3	3	2	6	2	1	2	1	2	5.2	39	163	55	20	1.47	2	1	
52	6	M	1	2	2	1	3	3	2	8	2	2	1	2	1	5.3	54	110	42	25	1.12	2	2	
53	7	M	2	1	2	2	2	2	2	6	1	2	1	1	2	4.2	60	125	37	28	1.18	2	2	
54	6	F	2	1	1	1	3	3	2	11	2	2	1	2	1	4.3	55	126	40	26	1.12	2	2	
55	8	M	2	1	2	2	2	2	2	6	1	2	1	1	2	3.9	51	160	46	24	1.12	2	2	
56	6	M	2	2	2	1	3	2	2	24	1	1	2	2	2	5.8	53	162	42	25	1.12	2	2	
57	9	M	2	1	2	2	2	2	2	1.45	2	2	1	2	2	4	54	140	40	25	1.12	2	2	
58	9	M	1	2	2	2	3	2	1	7	2	1	2	2	1	4.2	46	160	47	24	1.35	2	2	
59	7	M	2	1	2	2	3	2	2	9	2	2	1	2	1	4.3	50	134	51	24	1.18	2	2	
60	5	M	1	2	2	2	2	2	2	3	1	1	2	1	2	4.6	56	104	37	25	1.24	2	2	
61	4	M	2	2	2	2	2	2	2	11	2	2	1	1	2	4.7	53	126	35	25	1.12	2	2	
62	5	M	2	2	2	2	3	2	1	7	2	1	2	1	2	6	40	168	90	21	1.35	1	2	
63	6	M	1	2	1	2	2	2	2	4	2	2	1	1	2	4.8	49	160	66	23	1.12	2	2	UK
64	5	M	1	2	2	2	2	2	2	3	2	1	2	1	2	6	40	164	88	22	1.35	1	2	
65	5	M	1	2	2	2	3	2	2	12	1	1	2	2	1	4.3	44	160	49	22	1.12	2	2	
66	7	M	1	2	2	2	2	2	2	4	1	2	1	1	2	5	43	162	52	21	1.35	2	1	
67	4	M	1	1	2	2	3	3	2	6	4	1	1	2	2	4.8	42	164	54	21	1.35	2	2	
68	7	F	2	1	1	1	3	3	1	4.5	2	1	2	1	2	5.8	42	178	56	21	1.47	1	1	
69	6	M	1	1	2	2	3	2	2	2.5	1	1	2	2	1	3.8	40	164	48	21	1.24	2	2	
70	6	M	1	2	2	2	3	2	2	24	2	1	2	2	2	3.9	54	130	38	24	1.12	2	2	
71	5	M	2	2	2	2	3	2	2	2.45	1	1	2	1	2	4.7	53	137	47	25	1.59	2	2	
72	6	M	1	1	2	2	3	2	2	2.5	1	1	2	1	2	5.1	64	90	38	28	1.35	2	2	
73	6	M	1	1	2	1	2	3	2	5	4	2	1	1	2	5	47	140	42	27	1.12	2	2	
74	5	M	1	2	2	1	2	2	2	12	5	1	2	2	2	4.6	46	160	46	22	1.29	2	2	
75	8	M	2	2	1	2	2	2	2	5	4	2	1	1	2	4.9	39	140	42	26	1.24	2	2	
76	6	M	1	2	1	1	3	2	2	10	1	1	2	2	1	6.4	35	93	84	23	1.53	1		

S.No.	Age	Sex	Smoking	SHT	DM	HDL	LDL	TGL	CAD	Median delay in hours	Killip classification	Area of Infarct AWMI	Others	Thrombolysis successful	Unsuccessful	LVIDd	LVEF	EDV	ESV	FS	WMSI	LV clot	MR	TX/ Not Tx
77	7	M	2	2	1	1	3	2	2	11	5	1	2	2	1	5.5	52	160	45	22	1.29	2	2	
78	6	M	1	2	2	2	3	2	1	7	2	1	2	1	2	4	51	138	41	24	1.24	2	2	
79	7	M	2	2	1	2	2	2	2	7	1	2	1	2	1	4.2	49	135	50	24	1.18	2	2	
80	6	F	2	1	1	1	3	2	1	8	1	2	1	1	2	4.7	48	158	64	22	1.12	2	2	
81	7	M	2	1	1	1	3	2	2	10	2	1	2	2	1	6.6	32	171	92	22	1.53	1	2	
82	5	M	2	2	2	1	2	3	2	10	5	2	1	1	2	4.3	56	103	38	25	1.24	2	2	
83	7	M	2	2	2	1	3	3	2	4	2	2	1	1	2	4.2	30	96	35	18	1.24	2	2	
84	6	M	1	1	2	1	2	2	2	3	2	1	2	1	2	5.9	41	170	88	24	1.71	1	1	
85	6	M	1	1	2	2	3	2	2	6	4	1	2	2	1	5.8	42	92	84	23	1.53	1	2	
86	5	M	1	2	2	1	2	2	2	5.5	2	1	2	1	2	6.1	42	160	45	22	1.29	2	2	
87	4	M	1	2	2	2	3	2	2	8.5	1	2	1	1	2	4.9	61	90	38	28	1.35	2	2	
88	7	M	1	2	2	2	2	2	1	12	3	1	2	2	1	5	35	134	49	23	1.18	2	2	
89	6	M	1	2	2	2	3	2	2	7	1	1	2	1	2	4.5	54	140	41	27	1.24	2	2	
90	5	M	1	1	2	1	2	3	2	12	1	2	1	2	2	5.8	41	170	93	22	1.53	1	2	NT
91	6	M	1	2	1	1	3	2	2	6	1	2	1	2	1	6.4	43	136	37	24	1.24	2	2	
92	6	M	2	2	1	2	3	2	1	6.5	1	2	1	2	1	5.3	54	146	43	23	1.18	2	2	
93	6	M	2	2	1	2	2	2	2	12	2	1	2	2	2	5.8	41	142	79	24	1.59	1	2	NT
94	5	M	2	1	2	2	3	2	2	9	1	1	2	2	2	5.7	58	136	41	22	1.24	2	2	
95	4	M	1	2	2	1	3	2	2	2.15	2	1	2	2	2	4.8	50	146	44	23	1.18	2	2	
96	5	M	2	1	1	2	2	3	2	12	1	2	1	2	2	4.3	63	136	41	23	1.12	2	2	
97	7	M	1	2	2	2	3	2	2	6.5	1	1	1	2	2	4.6	49	130	42	22	1.24	2	2	
98	6	M	1	2	2	2	2	3	2	4.15	1	1	2	1	2	5.9	39	173	84	23	1.35	1		
99	7	M	2	2	2	2	3	3	2	14	1	1	2	2	2	3.7	56	131	37	24	1.24	2	2	
100	7	M	1	2	2	2	2	3	2	11.5	2	2	1	2	1	4.6	48	158	67	23	1.12	2	2	
101	4	M	1	2	1	2	2	2	2	5	2	1	2	1	2	4.7	34	161	52	21	1.35	2	2	
102	5	M	1	1	2	2	3	3	2	4.5	4	2	1	1	2	4.9	42	132	39	24	1.12	2	G2	
103	7	F	2	2	2	2	2	2	2	6.5	1	1	2	2	1	5.5	35	156	64	22	1.47	2	G2	

S.No.	Age	Sex	Smoking	SHT	DM	HDL	LDL	TGL	CAD	Median delay in hours	Killip classification	Area of Infarct AWMI	Others	Thrombolysis successful	Unsuccessful	LVIDd	LVEF	EDV	ESV	FS	WMSI	LV clot	MR	TX/ Not Tx
104	6	M	1	1	2	2	2	2	2	6.5	1	2	1	1	2	5.7	49	161	41	25	1.12	2	2	
105	5	M	1	2	2	2	3	3	2	5.5	2	1	2	1	2	6.2	38	156	62	21	1.59	1	G2	
106	6	M	1	2	2	2	3	2	2	4.5	1	1	2	1	2	3.3	55	164	36	24	1.24	2	2	
107	5	M	1	1	1	2	2	3	2	5	1	2	1	2	1	5.7	65	163	47	22	1.24	2	2	
108	8	M	2	2	2	2	2	2	2	9	1	2	1	2	1	5.1	40	160	46	21	1.24	2	2	
109	6	M	2	2	2	2	3	2	2	10	1	2	1	2	1	4.9	57	133	39	23	1.12	2	2	
110	4	M	1	2	2	2	3	2	2	7.5	1	1	2	1	2	5.8	49	130	53	24	1.18	1	2	
111	4	M	1	2	2	2	2	2	1	9	1	1	2	2	1	5.8	42	161	57	21	1.24	1	2	
112	8	F	2	2	2	2	3	2	2	3.5	2	1	2	2	1	4.3	53	139	53	24	1.18		2	
113	7	M	1	2	2	2	2	2	2	8	1	1	2	1	2	4.7	41	97	41	22	1.35	2	2	
114	6	M	2	2	2	2	2	2	2	6	1	1	2	2	2	5.9	36	95	78	24	1.41	1	G1	NT
115	7	M	2	2	2	2	2	2	2	24	5	1	2	2	2	7.5	39	98	83	23	1.35	1	2	NT
116	4	M	1	2	2	2	2	3	2	24	5	1	2	2	2	5.8	36	142	79	23	1.59	1	2	NT
117	5	M	1	2	2	2	3	2	2	10	1	1	2	2	1	4.6	37	87	40	22	1.35	2	2	
118	4	M	2	1	2	2	2	3	2	4	1	2	1	1	2	5.3	51	143	43	24	1.18	2	2	
119	5	M	2	2	2	2	3	2	2	3	2	1	2	1	2	5.8	41	140	80	23	1.59	1	2	
120	3	M	1	2	2	2	3	2	2	5	1	1	2	1	2	5.4	43	95	62	22	1.21	2	2	
121	7	F	1	2	2	2	3	2	2	4.5	1	2	1	1	2	4.9	60	137	40	23	1.12	2	2	
122	4	M	2	2	2	2	2	2	2		1	1	2	1	2	4.2	54	130	47	24	1.18	2	2	
123	6	M	2	2	2	2	3	2	2	5.5	1	1	2	2	2	4.7	46	86	46	24	1.24	2	2	
124	5	M	2	2	2	2	2	3	2	6.5	1	1	2	2	2	6.7	28	70	37	17	1.29	2	2	
125	5	M	1	2	2	2	2	2	2	3.5	1	1	2	2	2	5.1	44	96	64	23	1.24	2	2	
126	5	M	1	2	2	2	2	2	2	7	1	1	2	2	2	4.2	54	131	96	24	1.18	2	2	
127	5	M	2	2	2	2	2	2	2	4.5	1	1	2	1	2	4	52	137	43	24	1.24	2	2	
128	6	M	2	2	2	2	3	3	2	6	1	2	1	1	2	5	46	139	41	26	1.12	2	2	
129	5	M	1	1	2	2	2	2	2	24	1	1	2	2	2	5.8	38	141	81	26	1.24	1	2	NT
130	4	M	1	2	2	2	2	2	2	24	1	1	2	1	2	5.8	35	170	91	22	1.57	1	2	

S.No.	Age	Sex	Smoking	SHT	DM	HDL	LDL	TGL	CAD	Median delay in hours	Killip classification	Area of Infarct AWMI	Others	Thrombolysis successful	Unsuccessful	LVIDd	LVEF	EDV	ESV	FS	WMSI	LV clot	MR	TX/ Not Tx
131	5	M	1	1	2	2	3	2	2	4	2	1	2	1	2	5.4	42	98	37	25	1.24	2	2	
132	6	M	2	2	2	1	3	3	2	24	1	1	2	2	1	5.6	36	97	36	22	1.24	2	2	
133	5	M	1	2	2	2	2	2	2	4.5	2	1	2	1	2	4.6	42	87	37	29	1.35	2	2	
134	5	M	2	2	2	2	2	3	2	7	1	1	2	2	1	5.2	63	90	38	27	1.35	2	2	
135	6	M	2	1	2	2	2	2	2	4	1	1	2	1	2	4.8	41	141	49	22	1.24	2	2	
136	4	M	1	1	2	2	3	2	2	5.5	2	1	2	1	2	5.9	41	172	81	23	1.53	1	2	
137	5	M	2	2	1	2	3	2	2	7.5	1	1	2	2	1	5.4	53	157	44	22	1.29	2	2	
138	4	M	1	1	2	1	2	2	2	8	2	1	2	2	1	5.7	49	135	59	24	1.18	1	2	
139	5	M	2	2	2	2	2	3	2	3.5	1	1	2	1	2	3.9	52	137	42	24	1.24	2	2	
140	6	M	1	1	2	2	2	2	2	6	1	1	2	1	2	5.4	51	157	44	22	1.29	2	2	
141	4	M	2	2	2	2	3	2	2	7.5	2	1	2	1	2	4.6	56	104	37	25	1.24		2	
142	5	M	1	2	1	2	3	2	2	6.5	2	1	1	2	2	5.9	40	165	87	21	1.35	2	G1	
143	6	M	2	1	2	2	2	2	2	8	1	1	2	2	1	3.7	40	161	48	21	1.24		2	
144	5	M	1	2	2	1	3	3	1	5	2	1	2	1	2	6.2	38	175	93	21	1.47	2	2	

Abbreviations in Master Chart

Risk Factor
Present – 1
Absent - 2

Lipid Profile (LDL, HDL, TGL in mgs%)
If Decreased = 1
If Normal = 2
If Increased = 3

Normal HDL >50 in female ; >40 male
Normal LDL < 160 (if 1 or No risk factor) < 130 (2 or > risk factor)
< 100 if diabetes present
Normal TGL < 150

Thrombolysis successful = 1
Thrombolysis unsuccessful = 2
TX = Thrombolysed

LV clot present = 1
LV clot absent = 2